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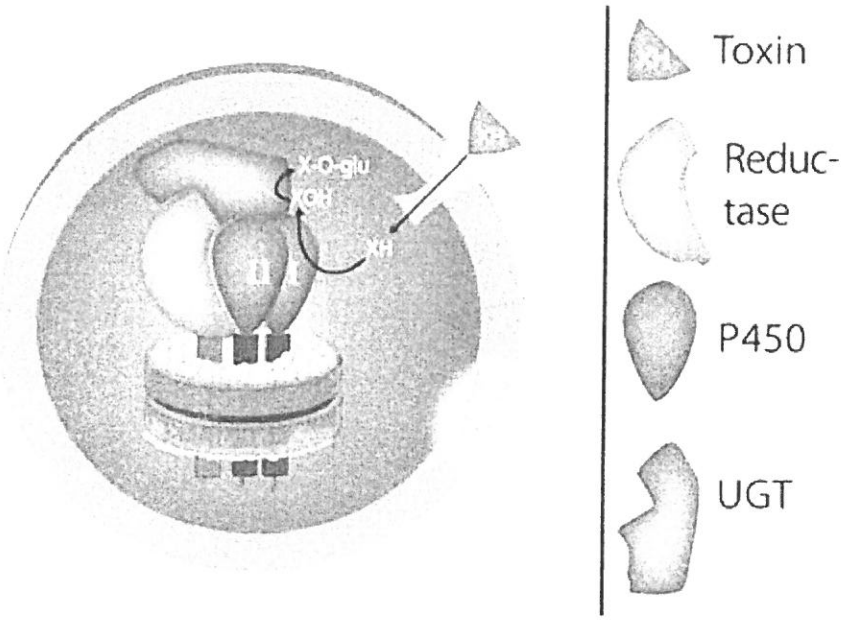

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Intricate interaction between pectins and pathogens

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The cell wall is one of the most important structural components of plants. The wall defines cell shapes, provides strength to withstand the turgor pressure and serves as the last physical barrier against invading pathogens.

Previous studies on a subset of cell wall mutants indicate that the cell wall polysaccharides contribute to defence not just as mechanical barriers but also as sensors for incoming infections. The *cev1* mutant of the model plant *Arabidopsis thaliana* has altered cellulose synthesis and shows enhanced resistance to necrotrophic pathogens due to upregulation of genes controlled by the defence-related signalling molecule jasmonic acid (Ellis et al., 2002). The *pmr4* mutant, which is mutated in a callose synthase, has enhanced resistance towards biotrophic pathogens due to hyper activation of the defence-related signalling molecule salicylic acid pathway (Nishimura et al., 2003). It appears that the cell wall integrity is tightly linked to the intracellular defence responses via mechanisms that may include yet uncharacterised sensory system(s).

We have recently identified a pectin mutant of *Arabidopsis thaliana*, *arabinan deficient 1 (arad1)*, defective in the pectic arabinan biosynthesis. Detailed cell wall composition analyses identified that *arad1* has 70% less arabinose in the pectic rhamonogalacturonan I fraction. The mutant did not show a visible growth phenotype distinct from the wild type, indicating that arabinan is not essential for plant growth. However, *arad1* mutants showed increased susceptibility to the necrotic fungal pathogen *Botrytis cinerea*, while they appear to show the wild-type level of susceptibility to the bacterial pathogen *Pseudomonas syringae*. Preliminary results suggest a possibility that arabinan may have a role in host defense due to a structural role in cell wall integrity. In addition, the identification of arabinan-degrading activity in *B. cinerea* suggests that the fungus has evolved mechanisms to overcome this barrier.

Exact molecular mechanisms responsible for the observed pathogen responses are currently under investigation and will be presented.